



**University of
Zurich**^{UZH}

**Zurich Open Repository and
Archive**

University of Zurich
University Library
Strickhofstrasse 39
CH-8057 Zurich
www.zora.uzh.ch

Year: 2013

Hyperadrenocorticism in dogs: diagnostic work-up according to the new ACVIM guideline

Reusch, Claudia E

Posted at the Zurich Open Repository and Archive, University of Zurich
ZORA URL: <https://doi.org/10.5167/uzh-81751>
Conference or Workshop Item

Originally published at:

Reusch, Claudia E (2013). Hyperadrenocorticism in dogs: diagnostic work-up according to the new ACVIM guideline. In: Annual meeting of the Portuguese Veterinary Small Animal Association, Lisbon, Portugal, 11 May 2013 - 12 May 2013.

Hyperadrenocorticism in dogs: diagnostic work-up according to the new ACVIM guideline

Claudia E. Reusch

Prof. Dr., Dipl ECVIM-CA

Clinic for Small Animal Internal Medicine, University of Zurich, Switzerland

In 2012 several endocrinologists from the US and Europe were asked by the ACVIM to form a Consensus Panel and to establish guidelines for the diagnosis of canine hyperadrenocorticism (HAC). The conclusions were presented during the ACVIM congress in June 2012, they will also be published in the near future. During the presentation, the principle of testing for HAC and the recommendations will be discussed.

Endocrine testing should only be performed when clinical signs consistent with HAC are present. The awareness of HAC by veterinarians has improved over the past 4 decades and dogs are currently evaluated for HAC at a much earlier stage in the disease development. Dogs may have one or more of the typical clinical signs; if only one clinical sign is present, it is most often polyuria and polydipsia or alopecia.

Clinical manifestations of HAC may also develop secondary to the space-occupying nature of a pituitary or adrenal tumor. Approximately 10-25% of dogs with pituitary-dependent HAC (PDH) develop neurologic signs due to a large tumor. An adrenocortical carcinoma may cause retroperitoneal hemorrhage or ascites or hind limb paresis due to tumor thrombus in the vena cava. The most common laboratory abnormalities are increased ALP, increased ALT, hypercholesterolemia, proteinuria and urine specific gravity in the hypo- or isosthenuric range. Dogs should not be tested for HAC as long as another serious disease exists, since illness may cause false positive results.

The diagnosis of HAC depends on the demonstration of two principals: 1) increased cortisol production and 2) decreased sensitivity to glucocorticoid feedback. Increased cortisol production is evaluated by the urinary corticoid: creatinine ratio (UCCR) and decreased feedback by means of the low-dose dexamethasone suppression test (LDDST). Those tests were introduced into veterinary medicine in the 1970's and 1980's, reference ranges and cut-off values were established at the time and are still in use. This fact is responsible for part of the diagnostic problems seen today. As the dogs are nowadays presented earlier as 20 or 30 years ago, milder cases will have lower levels of cortisol hypersecretion and test results may be borderline or even fall within the reference range.

The LDDST is performed by taking a zero blood sample, thereafter injecting 0.01 mg/kg dexamethasone iv and taking additional sample after 4 and 8 hours. The cortisol concentration after 8 hours is used for the diagnosis of HAC, however, it is important to note that resistance to dexamethasone is not an all or nothing phenomenon but a continuum. The reported sensitivity of the LDDST to diagnose HAC ranges from 85 to 100% and the reported specificity varies from 44 to 73%.

The Consensus Panel considers the LDDST the screening test of choice unless iatrogenic HAC is a possibility, however, cut-off values need to be redefined.

The UCCR reflects the corticoid production over several hours, thereby adjusting for fluctuations in cortisol blood levels. The reported sensitivity of the UCCR ranges from 75%-100% and the

reported specificity is 20-77%. In mild cases, a UCCR may be just within the reference range one day and elevated another. The urine sample should be taken at home by the owners to avoid any influence of stress. Due to cases of HAC being diagnosed in the early or mild stages, as for the other tests, the current cut-off values should also be re-evaluated.

The ACTH stimulation test is a test of adrenal reserve. It is the gold standard for diagnosis of iatrogenic HAC and adrenocortical insufficiency. The test has also been used as a screening test for spontaneous HAC. However, due to its low sensitivity, its diagnostic usefulness is lower than that of the LDDST.

To differentiate between the two major forms of HAC (PDH and AT) measurement of endogenous ACTH is the most accurate test. Reference ranges vary with technique and the correct sample handling is of utmost importance. The cortisol concentrations 4 hours after dexamethasone in the LDDS test support the presence of PDH if it is below the cut-off of the laboratory or < 50% of the basal cortisol concentration. Adrenal ultrasonography is helpful for the differentiation and can provide a good estimate for the presence of an adrenal tumor. However, an endocrine diagnosis cannot be based on diagnostic imaging, but requires hormone test.

Reference

ACVIM Forum Consensus Statement 2012. Diagnosis of Spontaneous Hyperadrenocorticism.
Ellen N. Behrend, Hans Kooistra, Richard Nelson, Claudia E. Reusch, Catharine Scott-Moncrieff